**PHYSIOLOGY ASS 16**

**ABU ANGEL ANONE**

**18/MHS02/006**

**NURSING SCIENCE (200l)**

**QUESTION :**

**Discuss Micturition**

**ANSWER :**

**MICTURITION CAN BE DEFINED AS THE PROCESS OF URINATION.**

**Micturition involves the simultaneous coordinated contraction of the bladder detrusor muscle, which is controlled by parasympathetic (cholinergic) nerves, and the relaxation of the bladder neck and sphincter, which are controlled by sympathetic (α-adrenergic) nerves.**

**Neuroanatomy**

**Micturition is dependent on complex interactions between the peripheral somatic and autonomic nerves, spinal cord, and brain. The bladder receives its motor innervation through the parasympathetic pelvic nerves. Its principal nuclei consist of motor neurons located in the gray matter of the intermediolateral cell column of the sacral spinal cord and motor neurons in the ventral gray matter of the sacral spinal cord in the region of Onuf's nucleus. In humans, sacral nerve blocks have revealed that the detrusor nucleus has a rostral–caudal extension going from the S3 to S4 segment. The precise intramedullary location of detrusor motor neurons and their histological characteristics in the sacral spinal cord have not been described. The bladder neck is innervated by the hypogastric nerves, which are derived from spinal segments T11–L2 (sympathetic). The external sphincter receives somatic innervation via a branch of the perineal nerve, the second branch of the pudendal nerve. Its motor neurons are located in Onuf's nucleus, in spinal segments S2–S4.**

**The sensory innervation of the autonomically innervated structures is presumed to be through the corresponding visceral afferents. The urethra distal to the external sphincter receives sensory innervation from branches of the pudendal nerve.**

**The areas of the central nervous system specifically defined in bladder innervation include the cerebral cortex, the pons, and the conus medullaris. The principal afferent and efferent spinal pathways from detrusor motor neurons in the conus medullaris have been identified in the posterior superficial portion of the lateral columns. The spinal pathways for innervation of the external urinary sphincter have not been identified in humans. However, they can be assumed to resemble the organization of skeletal muscle, with ascending pathways in the posterior columns and descending tracts in the corticospinal and reticulospinal pathways.**

**Areas near the nucleus locus ceruleus in the pons have been identified as crucial for the appearance of reflex detrusor contractions in certain quadriped animals. The location of the pontine nucleus responsible for detrusor contractions (also known as the pontine micturition center) in humans has not been identified but is presumed to be similar to that in animals, although reports of bladder dysfunction in humans with pontine lesions are limited. There are bilateral descending projections from homologous pontine nuclei to the intermediolateral cell column of the sacral spinal cord, through which efferent impulses travel to initiate a detrusor contraction.**

**The cerebral cortex, particularly areas of the prefrontal cortex, has been identified as having regulatory influences on detrusor function. Patients with lesions of the prefrontal cortex are unable to suppress detrusor contractions (detrusor overactivity), suggesting that cortical (volitional) control of bladder function is particularly dependent on this portion of the brain. Other brain areas that have been demonstrated to influence bladder function in animals and humans include the periaqueductal gray, insula, anterior cingulate cortex, insula, basal ganglia, thalamus, hypothalamus, limbic system, and cerebellum.**

**Physiology**

**The micturition cycle involves two phases: bladder filling/urine storage and bladder emptying. Bladder filling requires (i) accommodation of increasing volumes at low intravesical pressure (compliance) and appropriate sensation, (ii) the bladder outlet to be closed at rest and during increases in intra-abdominal pressure, and (iii) the absence of involuntary bladder contractions. Bladder emptying requires (i) coordinated contraction of the bladder of adequate magnitude and duration, (ii) lowering of resistance at sphincters, and (iii) the absence of obstruction (e.g., enlargement of the prostate gland in the aging male may result in bladder outlet obstruction, precluding efficient micturition). Bladder dysfunction can then be clinically identified as a problem of filling or emptying or a combination of both, and the site of dysfunction may be the bladder, the urethra, or both.**

**The normal function of the bladder is to store urine until it has reached capacity and until it is socially acceptable to evacuate urine. Urine storage is accomplished at low pressures, measured as compliance. Compliance is calculated as the change in volume over the change in pressure. Bladder compliance is a result of the viscoelastic properties of the bladder. The bladder wall contains elastin, which allows it to stretch without a subsequent increase in pressure. Typical adult bladder capacity is approximately 350–450 ml. Compliance is also facilitated by sympathetic discharge primarily mediated through the β-adrenergic receptors within the bladder wall. This sympathetic tone operates directly at the level of the bladder musculature to facilitate storage. There is also sympathetic discharge at the level of the autonomic ganglia, which has an inhibitory effect on the parasympathetic postganglionic neurons, thus preventing detrusor contraction and facilitating urine storage. Loss of compliance may lead to renal insufficiency.**

**Continence is maintained through the action of the urinary sphincters. The internal sphincter, or the bladder neck, is richly innervated with α-adrenergic receptors. During bladder filling this structure remains closed through constant sympathetic discharge via the hypogastric plexus. The external sphincter, composed of striated muscle, also maintains a resting tone to maintain continence. It is believed that the fibers of the external sphincter are primarily of the slow twitch variety and thus can maintain tension for long periods of time. With rapid increases in intra-abdominal pressure, fast twitch fibers are recruited to contract and further increase the urethral resistance to avoid urinary leakage.**

**As the bladder fills, its visceral afferents travel through the peripheral nerves, ascending through the spinal cord to the pontine micturition center. It is at this level that a detrusor contraction is initiated. However, there are inhibitory signals from suprapontine centers (e.g., prefrontal cortex and basal ganglia) that prevent the generation of a detrusor contraction until the bladder is full. At normal bladder capacity (350–450 ml), sensations of fullness are transmitted through detrusor afferents, nerves that provide reflex excitation through the central nervous system to the motor innervation to the detrusor. The cortex releases its inhibition of the pontine micturition center. An efferent response travels through the spinal cord and through the pelvic nerves, and a detrusor contraction occurs. The contraction is coordinated with the opening of the internal and external sphincters to allow free egress of urine outside the body. A normal detrusor contraction is of adequate strength and duration to empty the bladder completely in one coordinated contraction. After urine evacuation, the sphincters return to their closed state and the cycle resumes.**